IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF OHIO

LAUREN BOSSETTI, individually and as mother and next friend of S.R. Bossetti, - And - DEBORAH DIMEGLIO, individually and as mother and next friend of L.D., - And - TARA GUIDA, individually and as mother and next friend of V.P. Guida, Plaintiffs, v. ALLERGAN SALES, LLC. **Serve:** **Corporate Creations Network Inc.** **119 E. Court Street** **Cincinnati, OH 45202** Defendant.	Case No.:

COMPLAINT

NOW COMES the Plaintiffs by and through her attorneys, states as follows:

PARTIES AND JURISDICTION

1. Plaintiff Lauren Bossetti is a citizen and resident of Huron County in the State of Ohio. S.R. Bossetti was born on August 19, 2012. Plaintiff alleges that Defendant's Lexapro® was defectively designed, inadequately tested, dangerous to human health and unborn, and lacked proper warnings as to the true danger associated with its use, and S.R. Bossetti suffered injury as a result of Plaintiff Bossetti's ingestion of Lexapro®.

2. Plaintiff Deborah DiMeglio is a citizen and resident of Mercer County in the State of New Jersey. L.D. was born on December 7, 2009. Plaintiff alleges that Defendant's Lexapro® was defectively designed, inadequately tested, dangerous to human health and unborn, and lacked proper warnings as to the true danger associated with its use, and L.D. suffered injury as a result of Plaintiff DiMeglio's ingestion of Lexapro®.

3. Plaintiff Tara Guida is a citizen and resident of Virginia Beach County in the State of Virginia. V.P. Guida was born on September 20, 2010. Plaintiff alleges that Defendant's Lexapro® was defectively designed, inadequately tested, dangerous to human health and unborn, and lacked proper warnings as to the true danger associated with its use, and V.P. Guida suffered injury as a result of Plaintiff Guida's ingestion of Lexapro®.

4. At all times relevant to this Complaint, Defendant Allergan Sales, LLC was and is the successor in liability for sale of any Lexapro® that could have caused any Plaintiff's injury. Allergan Sales, LLC is diverse from the plaintiffs. Its members are Allergan Holdco US, Inc., and Allergan Holdings, Inc., each of which is a Delaware corporation. Allergan Holdco US, Inc., and Allergan Holdings, Inc., each have its principal place of business in California.

STATEMENT OF FACTS

5. Each of the preceding paragraphs is incorporated by reference herein.

6. At all relevant times, Defendant was in the business of and did design, research, manufacture, test, advertise, promote, market, sell, distribute, and/or have acquired and are responsible for entities which have designed, researched, manufactured, tested, advertised, promoted, marketed, sold and distributed the pharmaceutical drug Lexapro®.

7. Each Plaintiff was prescribed Lexapro® and used it as directed.

8. Lexapro® is an antidepressant and anti-anxiety drug belonging to the group of selective-serotonin reuptake inhibitors ("SSRI" or "SSRIs").

9. Lexapro® was invented and developed for use as an antidepressant by a predecessor to Allergan Sales.

10. According to the Centers for Disease Control, during 2008–2013, on average, 15.4% of reproductive-aged women (range = 15.3%–15.6%) filled a prescription for an antidepressant from an outpatient pharmacy each year.

11. Lexapro® is the S-stereoisomer of its predecessor drug, Celexa®, which is also an SSRI manufactured and marketed by Defendant.

12. Though Lundbeck developed Lexapro®, Lundbeck partnered with one of Defendant's predecessors to manufacture, market, and distribute the drug, with the two sharing in the revenue.

13. Lexapro® was one of Forest Laboratories' biggest revenue making drugs with total sales approaching 3 billion dollars in 2008.

14. SSRIs and the related serotonin–norepinephrine reuptake inhibitors ("SNRIs") are the most prescribed antidepressants in use.

15. The first SSRI entering medical use was fluoxetine, known by the brand name Prozac® ("Prozac").

16. Prozac, developed by Eli Lilly in 1972, entered medical use in 1986 and the U.S. FDA gave it final approval in 1987.

17. In the brain, messages are passed between two nerve cells via a chemical synapse, a small gap between the cells. The (presynaptic) cell that sends the information releases neurotransmitters (including serotonin) into that gap.

18. The neurotransmitters are then recognized by receptors on the surface of the recipient (postsynaptic) cell, which upon this stimulation, in turn, relays the signal.

19. About 10% of the neurotransmitters are lost in this process; the other 90% are released from the receptors and taken up again by monoamine transporters into the sending (presynaptic) cell (a process called reuptake).

20. SSRIs inhibit the reuptake of serotonin ("5-hydroxytryptamine" or "5-HT"). As a result, the serotonin stays in the synaptic gap longer than it normally would, and may repeatedly stimulate the receptors of the recipient cell.

21. Levels of 5-HT are regulated by the serotonin transporter ("SERT") which is the target of SSRIs.

22. It is generally accepted that 5-HT serves a critical role in neurodevelopment.

23. 5-HT modulates a variety of processes during neurodevelopment and is thus disruptive to the 5-HT levels during neurodevelopment can deregulate a number of developmental processes leading to long-term damage to the brain and its neural networks.

24. This critical development period begins during a developing child's embryonic stage and can continue into early infancy.

25. Interference with development during this critical period can lead to long-term biological and biochemical deficits that are manifest in a variety of clinical symptoms.

26. This disruption in the biochemical environment the womb can lead to such significant alterations in biochemical and neurotransmitter identity, density, and arrangement, such that the developed child exhibits behaviors that are not "typical" of non-disrupted neurological development.

27. SSRI administration can lead to alterations in 5-HT homeostasis during development.

ANIMAL EVIDENCE OF SSRI IMPACTS ON TYPICAL NEURODEVELOPMENT

28. Adult mice using SSRIs at therapeutically relevant doses during the equivalent of the third trimester of human development produced a similar abnormality to the adult mice as those seen with genetic SERT deletion.

29. Neonatal exposure of SSRI antidepressants to rats has been repeatedly shown to result in sensory and social abnormalities that parallel those reported in autism spectrum disorders in humans.

30. Studies done in rats demonstrate that citalopram exposure to rats during the sensitive period of brain development resulted in long-lasting alterations in the norepinephrine locus coeruleus neural activity, providing evidence of connections between SSRI exposures to human developmental disorders.

31. Since the first animal studies were done, a number of studies have drawn conclusions that echo the sentiment that there may be significant behavioral consequences of antidepressant use during pregnancy.

SEROTONIN'S RELEVANCE TO NEUROLOGICAL ABNOMALITIES IN CHILDREN

32. In 1961, Schain and Freedman first reported hyperserotonemia, a condition where serotonin levels are disrupted and elevated in the blood plasma, in autistic patients.

33. This has been followed by multiple similar studies finding that between one-fourth and one-third of autistic people exhibit serotonin disturbances in the form of hyperserotonemia.

34. In 1995, McBride *et. al.* reported that several studies have shown a positive correlation of platelet serotonin levels between probands with autism and their parents and siblings.

35. Correlations were also found between platelet serotonin levels and the rate of platelet serotonin transport.

36. By 1990, the critical role of serotonin in neural distortions became clear to medical and biochemical researchers, including those within the pharmaceutical industry, and thus the pharmaceutical industry began to study and report the clinical effectiveness of treating patients with these disruptions with SSRI drugs.

37. Potent serotonin transporter inhibitors including SSRIs have been shown to reduce ritualistic behaviors associated with anxiety and to reduce aggression in more than 50% of children and adults with autism in both open and double-blind studies.

38. Chronic administration of SSRIs in rodents leads to an increase in neurotransmission through downregulation of presynaptic terminal autoreceptors.

39. Further evidence of serotonin's involvement in neurological developmental issues, including autism spectrum disorders, comes from a pharmacologic study using tryptophan depletion, which leads to reduced serotonin synthesis, release, and neurotransmission. After tryptophan deletion, researchers found exacerbation of neuro-atypical behaviors such as whirling, flapping, pacing, banging and hitting self, rocking, toe walking, and anxiety in a majority of adults with autism after tryptophan depletion.

40. Researchers thus postulated in 1996 that serotonin may have a role in the developmental neuropathologic abnormalities found in the hippocampus, amygdala, and cerebellum in people exhibiting autism spectrum disorders.

41. In 1999, Chugani *et. al.* found that autistic children produced far less serotonin in the brain than normal children.

42. In 2005, the International Journal of Developmental Neuroscience published an article titled "Behavioral and cellular consequences of increasing serotonergic activity during brain development: a role in autism?" detailing the potential mechanism by which disruption of serotonin homeostasis during neurodevelopment may lead to cellular changes responsible for behavioral abnormalities associated with autism.[1]

43. In 2011, Pat Levitt, PhD, summed up the significance of the interplay between SSRIs, serotonin and neurodevelopment in the Archives of General Psychiatry:[2]

> **The basic science data are rock solid in reflecting a clear role for 5-HT in developmental programming, particularly from studies in which 5-HT signaling such as disruption of the 5HT1A receptor is manipulated only during development. Enduring behavioral outcomes are readily measured. Excessive 5-HT in the brain due to monoamine oxidase A genetic deletion can alter the patterning of forebrain circuits. Furthermore, it has been shown that 5-HT can modulate the response of growing axons to important axon guidance molecules, again with the potential to affect wiring. Yet, perhaps the strongest connection lies in conceptually important features of the influence of 5-HT on fetal brain development: (1) the placenta, and not the mother, is the major source of 5-HT, with the synthetic capacity in both humans and mice to produce 5-HT from the maternally supplied essential amino acid tryptophan; (2) the fetal forebrain, but not the hindbrain, accumulates placental 5-HT, which occurs only for a limited period in mice that is equivalent to the first trimester in the human; and (3) because the placenta and fetus are genetically identical, the ascribed direct impact of mutations on the fetal brain may in fact be due to more complex maternal-placental-fetal interactions that underlie pathogenesis.**

[1] Patricia M Whitaker-Azmitia, *Behavioral and cellular consequences of increasing serotonergic activity during brain development: a role in autism?* International Journal of Developmental Neuroscience 75–83 (2005).
[2] Pat Levitt, *Serotonin and the autisms: a red flag or a red herring?* 68 Arch Gen Psychiatry 1093–1094 (2011).

THE RELEVANCE OF SSRI USE DURING PREGNANCY

44. SSRIs and their metabolites have been known for decades, and at least as early as 1989, to cross the placenta and enter the womb. One of the first studies done using rats was published in 1989 and concluded:[3]

> Visual and quantitative evaluations of the autoradiograms indicated that the highest fetal concentrations of radiocarbon were associated with brain and thymus. Results from these studies indicate that fluoxetine and norfluoxetine [SSRIs] traverse the placenta and distribute within the embryo/fetus during the periods of organogenesis and postorganogenesis and confirm embryonic/fetal exposure of parent and metabolite in previous negative rat teratology and reproductive studies.

45. Additional studies confirmed this in humans, finding that "[c]italopram, fluoxetine and desmethylfluoxetine all cross the human placenta, and may, therefore, affect the perinatal outcome of infants exposed to these drugs during pregnancy."

46. Evidence establishes that both SSRIs and SNRIs cross the placenta and lead to exposure to the developing fetus.

47. Furthermore, children born to women taking SSRIs during the later stages of pregnancy suffer from SSRI withdrawal, serotonin toxicity and poor adaptation effects as a result of serotonin disruption during pregnancy.

NEUROLOGICAL STUDIES CONFIRM EARLY DEVELOPMENTAL ETIOLOGY OF BEHAVIORAL ABNORMALITIES AMONG THOSE DIAGNOSED WITH AUTISM AND ASD

48. Starting at least as early as 1994, there has been mounting evidence that behavioral abnormalities that are associated with autism and ASD are the result of mal-development during early brain development in the womb.

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[3] R C Pohland et al., *Placental transfer and fetal distribution of fluoxetine in the rat*, 98 Toxicology and Applied Pharmacology 198–205 (1989).

49. For example, Courchesne et. al. published in early 1994 based on diagnostic scans of autistic brains that there were significant biological differences in the autistic brains which the authors deemed to be neuroanatomic maldevelopment that may have been triggered as early as the second trimester.[4]

50. Evidence of the patterns of neurological maldevelopment continued to surface with the work done by Courchesne et. al. This work highlights changes in neural density, brain formation and growth, the relative amounts of white and grey matter in the brain, and a number of other changes that are set in motion during neurodevelopment in the womb.

51. By 2004, Courchesne et. al. had concluded that "from current evidence, autism must be caused by prenatal or early postnatal events."[5]

52. The evidence of neural maldevelopment, occurring at the earliest stages of brain development, is repeated in study upon study in the medical literature.[6]

53. The evidence of developmental changes in the cellular architecture of the brain is not limited to the work done by Courchesne et al.

[4] E Courchesne, J Townsend & O Saitoh, *The brain in infantile autism: posterior fossa structures are abnormal*, 44 Neurology 214–223 (1994).
[5] Eric Courchesne, Elizabeth Redcay & Daniel P Kennedy, *The autistic brain: birth through adulthood*, 17 Current Opinion in Neurology 489–496 (2004).
[6] E Courchesne, J Townsend & O Saitoh, *The brain in infantile autism: posterior fossa structures are abnormal*, 44 Neurology 214–223 (1994); E Courchesne et al., *Unusual brain growth patterns in early life in patients with autistic disorder: an MRI study*, 57 Neurology 245–254 (2001); Eric Courchesne, Elizabeth Redcay & Daniel P Kennedy, *The autistic brain: birth through adulthood*, 17 Current Opinion in Neurology 489–496 (2004); Emanuel DiCicco-Bloom et al., *The developmental neurobiology of autism spectrum disorder*, 26 J. Neurosci. 6897–6906 (2006); Rich Stoner et al., *Patches of Disorganization in the Neocortex of Children with Autism*, 370 N. Engl. J. Med. 1209–1219 (2014).

54. In 2003 another group evaluated evidence of brain differences in children with autism finding differences in the biochemical and cellular cytoarchitecture of the brains of children with ASD.[7]

55. The past three or more decades of research on autistic brains confirms that behavioral abnormalities associated with ASD and autism are triggered by maldevelopment early in the process of assembly of the developing brain.

EPIDEMIOLOGICAL EVIDENCE CONFIRMS ANIMAL STUDIES AND HUMAN BIOCHEMICAL AND NEURODEVELOPMENTAL EVIDENCE THAT SSRI USE DURING PRENGNACY DISTRUPTS BRAIN DEVELOPMENT RESULTING IN BEHAVIORAL ABNORMALITIES

56. The combined understanding of the critical role of serotonin in brain development, knowledge that SSRIs cross the placenta and work to disrupt normal brain development, the current understanding of the maldevelopment and derangement in the brains of those with ASD and autism, along with the demonstration through animal studies of cause and effect, provide ample evidence of both a plausible mechanism and the probability of the outcome:

> **that the use of SSRIs during pregnancy crosses the placenta, disrupts serotonin homeostasis in the developing embryo at a critical time of the chemical and structural development of the brain, leading to maldevelopments in the human cellular and biochemical anatomy that result in behavioral abnormalities in the developed child.**

57. These suggestions are further confirmed by human epidemiological studies of children born to mothers taking SSRIs.

58. A 2011 study by Croen et. al. found a 2-fold increased risk of ASD associated with treatment with SSRIs by the mother during the year before delivery with the strongest association

[7] S D Friedman et al., *Regional brain chemical alterations in young children with autism spectrum disorder*, 60 Neurology 100–107 (2003).

being with treatment during the first trimester, where the adjusted odds ratio was almost four-fold increased risk.[8]

59. The evidence of neurodevelopmental harm prompted Prescrire to publish an article recommending that SSRIs only be used during pregnancy when non-drug measures fail and when symptoms are sufficiently serious to warrant drug therapy.[9]

60. In 2013 Rai etc. all published an article in the British Medical Journal supporting this association. The researchers concluded that in utero exposure to SSRIs were associated with an increased risk of autism spectrum disorders.[10]

61. In 2014, Harrington et. al. published their findings in the journal Pediatrics. The results included the finding of a nearly 3-fold increased risk of ASD in boys born to mothers exposed to SSRIs, with the strongest association occurring in the first trimester (OR: 3.22; 95% CI: 1.17-8.84).[11]

62. A meta-analysis of studies written by Man et. al. was published in the journal Neuroscience and Biobehavioral Reviews. The meta-analysis found support for an increased risk of ASD in mothers exposed to SSRIs during pregnancy.[12]

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[8] Lisa A Croen, *Antidepressant Use During Pregnancy and Childhood Autism Spectrum Disorders*, 68 Arch Gen Psychiatry 1104–1112 (2011).
[9] SSRI antidepressants: altered psychomotor development following exposure in utero? *SSRI antidepressants: altered psychomotor development following exposure in utero?* 22 Prescrire Int 43–44 (2013).
[10] Dheeraj Rai et al., *Parental depression, maternal antidepressant use during pregnancy, and risk of autism spectrum disorders: population based case-control study*, 346 BMJ 2059–2059 (2013).
[11] Rebecca A Harrington et al., *Prenatal SSRI Use and Offspring With Autism Spectrum Disorder or Developmental Delay*, 133 PEDIATRICS e1241–e1248 (2014).
[12] Kenneth K C Man et al., *Exposure to selective serotonin reuptake inhibitors during pregnancy and risk of autism spectrum disorder in children: A systematic review and meta-analysis of observational studies*, 49 Neuroscience & Biobehavioral Reviews 82–89 (2015).

63. Providing further evidence of this connection, Boukhris et. al. published an article in JAMA Pediatrics in 2015 examining the risk of ASD in children born to mothers taking antidepressants. This paper concluded that the use of antidepressants, particularly SSRIs, during portions of pregnancy increase the risk of ASD.[13]

DEFENDANT'S INADEQUATE LABEL FAILED TO WARN OF RISKS THAT WERE KNOWN AND THOSE THAT SHOULD HAVE BEEN KNOWN AT THE TIME OF USE

64. One tragic aspect of the inadequate label is that Lexapro® causes irreversible and devastating injuries to the developing child before the mother or the physician even have a chance to discover the pregnancy. Defendant knew or should have known it had a duty to warn doctors and patients that women who were taking Lexapro® should not get pregnant, and that women who might become pregnant should not take Lexapro®. This simple warning, commonplace with countless pharmaceuticals, would have spared each Plaintiff a lifetime of pain and suffering, isolation, inordinate healthcare costs, severe emotional and physical distress, and loss of earning potential.

65. Lexapro® was and is a defective product, unreasonably dangerous in light of its nature and intended use. That defect existed when the product left Defendant's control and has been the proximate cause of injuries to Plaintiffs, whose injuries were caused by the use of Lexapro® in its intended or foreseeable manner or in the manner recommended by Defendant.

66. Defendant knew or should have known of the dangerous condition of its product, Lexapro®, but failed to adequately warn or instruct physicians and consumers of the risks, dangers, and proper uses of the drug.

[13] Takoua Boukhris et al., *Antidepressant Use During Pregnancy and the Risk of Autism Spectrum Disorder in Children*, JAMA Pediatr 1–8 (2015).

67. Defendant knew or should have known the critical role of serotonin in the development of the human brain and the risk of long-term impacts, including the causation of behavioral abnormalities, when serotonin levels are disrupted during development.

68. Defendant knew or should have known that Lexapro® and its metabolites would cross the placenta and disrupt that sacred homeostasis of serotonin levels in the womb, so critical to normal human development. Furthermore, Defendant knew or should have known that the disruption of this critical balance risks maldevelopment of the human brain in such a way as to cause behavioral abnormalities in the developed child.

69. Defendant has breached its duty of reasonable care and its express and implied warranties, and has made affirmative misrepresentations as well as misrepresentations by omission, all in connection with the design, testing, manufacture, marketing, and/or labeling of Lexapro®.

70. As a direct and proximate result of the acts and omissions of Defendant, the Plaintiffs' children were born with damaged brains manifest as severe behavioral, neurological and emotional injuries. Plaintiffs' children continue to suffer permanent injury, loss of normal life, and other non-economic damages

71. As a direct and proximate result of the aforesaid acts of and/or omissions by the Defendant, each child has:

(a) suffered severe and permanent injuries, which he will be forced to endure for the remainder of her life;

(b) suffered physical pain and suffering;

(c) suffered mental pain and suffering;

(d) suffered loss of enjoyment of life;

(e) incurred substantial costs for medical care in the past, and will in

reasonable medical probability incur substantial costs for medical care in the future;

(f) suffered a loss of earnings and of future earning capacity; and,

(g) incurred attorney's fees and expenses of litigation related to this action.

COUNT I
Strict Liability

75. Plaintiffs incorporate by reference all preceding paragraphs of this Complaint as if fully set forth herein, and further allege:

76. At all times relevant, Defendant had a duty to manufacture, test, market, advertise, label, distribute, and sell Lexapro® so that it was reasonably safe for its foreseeable use.

77. Due to design defects, at the time Lexapro® left the control of Defendant and was sold, it contained one or more conditions that rendered it defective and unreasonably dangerous in light of its nature and intended use.

78. The Lexapro® manufactured and/or supplied by Defendant and to which Plaintiffs were exposed was defective in design, and/or formulation in that when it left the hands of Defendant, the foreseeable risks (particularly risks of birth defects, congenital malformations, cognitive impairment, and neurobehavioral disorders to unborn children) exceeded the benefits associated with the design and/or formulation of this product.

79. The Lexapro® manufactured and/or supplied by Defendant was defective in design and/or formulation in that it was more dangerous than an ordinary consumer would expect when used in its intended or reasonably foreseeable manner.

80. The dangers presented by Lexapro® are so great that reasonable health care professionals would not prescribe its use by pregnant women or women who may become pregnant if they knew of the risks.

81. The dangers presented by Lexapro® are so great that reasonable consumers—such as Plaintiffs—would not use Lexapro® when they were pregnant or might become pregnant if they knew of the risks.

82. At all times, Plaintiff used Lexapro® in the manner intended, recommended, or reasonably foreseeable by Defendant particularly based on Lexapro® 's indications and/or Defendant's marketing of Lexapro®.

83. There were and are no other reasonable, secondary causes of Plaintiffs' injuries and damages other than the use of Lexapro®.

84. Defendant is strictly liable for injuries resulting from the defective design of their product.

85. As a direct and proximate result of Defendant wrongful actions, Plaintiffs suffer from permanent, profound, and debilitating conditions including pervasive developmental disorder and autism.

COUNT II
Negligence

86. Plaintiffs incorporate by reference all preceding paragraphs of this Complaint as if fully set forth herein, and further allege.

87. Defendant owed Plaintiffs and all consumers a duty of reasonable care in how they designed Lexapro®, manufactured Lexapro®, tested Lexapro®, and warned of Lexapro® dangers.

88. Defendant breached their duty of care by designing, manufacturing, testing, and labeling Lexapro® in a manner that was dangerous to women who were pregnant or might become pregnant.

89. A reasonable manufacturer would or should have known that Lexapro® 's risks are unreasonably greater than necessary.

90. The dangers presented by Lexapro® are so great that reasonable health care professionals would not prescribe its use by pregnant women or women who may become pregnant if they knew of the risks.

91. As a direct and proximate result of Defendant's wrongful actions, Plaintiffs' minor children suffer from permanent, profound, and debilitating conditions including pervasive developmental disorder autism.

COUNT III
Breach of Implied Warranty

92. Plaintiffs incorporate the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

93. Defendant was a merchant seller with respect to Lexapro.

94. In order to induce the purchase and/or use of Lexapro, Defendant impliedly warranted to potential users of Lexapro®, including Plaintiff and her medical providers, that Lexapro® was safely tested and manufactured and was safe for the uses for which it was designed and/or advertised to be used, including the use by women of childbearing age, those seeking to become pregnant, and those pregnant.

95. Defendant breached this warranty in that Lexapro® was not safe for the uses for which it was manufactured and/or advertised, particularly it was not safe for use by pregnant women or those seeking to become pregnant. The lack of safety is the result, at a minimum, of the damage that Lexapro® is capable of causing to the developing brain during pregnancy by disruption of serotonin levels within the developing ecosystem.

96. Furthermore, Defendant failed to provide an adequate warning in an attempt to remedy its defective product. Such warning would have highlighted the risks posed by the use of

Lexapro® during pregnancy and its ability to modify serotonin levels and disrupt the normal development of the brain, thereby causing irreversible brain damage.

97. Plaintiffs were injured as a result of detrimental reliance upon Defendant's implied warranties.

98. As a direct and proximate result of one or more of the foregoing breaches of implied warranty, Plaintiff suffered injuries and damages as described herein.

COUNT IV
Breach of Express Warranty

99. Plaintiffs incorporate the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

100. Plaintiffs incorporate the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

101. Defendant was a merchant and seller with respect to Lexapro.

102. In order to induce the purchase and/or use of Lexapro, Defendant expressly warranted to potential users of Lexapro®, including the mother Plaintiffs and their medical providers, that Lexapro® was safely tested and manufactured and was safe for the uses for which it was designed and/or advertised to be used including the use by women of childbearing age, those seeking to become pregnant, and those pregnant. Express warranties were contained in direct-to-consumer advertising and other promotional and marketing campaigns, Lexapro® product information sheets given to patients with their prescriptions, and other public communications and representations which indicated the product's safety for use during pregnancy and in women of childbearing age, including those intending to become pregnant.

103. Defendant breached said warranty in that Lexapro® was not safe to be used for the purposes for which it was manufactured and/or advertised, particularly it was not safe for use by pregnant women or those seeking to become pregnant.

104. Plaintiffs were injured as a result of detrimental reliance upon Defendant's express warranties.

105. As a direct and proximate result of one or more of the foregoing breaches of express warranty, Plaintiff suffered injuries and damages as described herein.

COUNT V
Failure to Warn

106. Plaintiffs incorporate the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

107. Defendant had a duty to warn of dangers known or that it should have known of, including that ingestion of Lexapro by pregnant mothers may increase the risk of neurodevelopmental delays and other cognitive defects.

108. As a direct and proximate cause of Defendant's failure to warn, Plaintiffs each ingested Lexapro while pregnant and their minor children suffered severe and permanent injuries as indicated.

COUNT VI
Strict Products Liability Design Defect O.R.C. § 2307.75

109. Plaintiff Bossetti brings this Count in the alternative to her common law claims.

110. Plaintiff incorporates the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

111. Defendant is the manufacturer, designer, marketer, distributor and sellers of Lexapro®.

112. The Lexapro® manufactured, designed, marketed, distributed and sold by Defendant was expected to and did reach the consumer without any alterations or changes.

113. The Lexapro® manufactured, designed, marketed, distributed and sold by Defendant was defective in design or formulation, because when it left the hands of Defendant, the foreseeable risks of the product exceeded the benefits associated with its design or formulation.

114. The Lexapro® manufactured, designed, marketed, distributed and sold by Defendant was defective in design or formulation, because when it left the hands of the Defendant, it was more dangerous than an ordinary consumer would expect.

115. The foreseeable risks of Lexapro® include an increase in the occurrence of major congenital malformations from fetal exposure to Lexapro®, the magnitude of which is dramatic in terms of the number of women exposed, the incidence rate, and the devastating harm to the fetus.

116. The fact that harm such as that suffered by S.R. Bossetti will occur from use of Lexapro® is completely foreseeable; Defendant has not prohibited Lexapro®'s use in women of childbearing years; half of all pregnancies in the United States are unplanned, and few contraception measures are 100% effective.

117. Lexapro® as manufactured, designed, marketed, distributed and sold by Defendant is much more dangerous than an ordinary consumer would expect, as maternal use of Lexapro® during fetal development creates a very high risk of cognitive, developmental, neurological and behavioral dysfunction and failed to provide adequate warning and instruction concerning the unavoidably unsafe aspect of Lexapro®.

118. At the time Defendant manufactured, designed, marketed, distributed and sold Lexapro® to Plaintiff, safer, more practical, alternative designs were available to treat depression.

119. The Lexapro® manufactured, designed, marketed, distributed and sold by Defendant was not unavoidably unsafe, as alternative formulations for anti-depressant medications were available with comparable or adequate efficacy that did not pose the same risk.

120. Based upon the foregoing, the Lexapro® manufactured, designed, marketed, distributed and sold by Defendants was defective in design pursuant to O.R.C. § 2307.75 at the time it left Defendants' control.

121. As a direct and proximate result of the defective design of Lexapro® consumed by Plaintiff, Plaintiff suffered damages, including but not limited to personal injury, bodily harm, emotional distress, pain and suffering, permanent physical, mental, neurologic, cognitive and behavioral injuries, loss of enjoyment of life, economic and non-economic damages, and will continue to suffer such injuries, distress, pain and suffering, harm,damages, and economic loss in the future.

122. In addition, as a direct and proximate result of the defective design of Lexapro® consumed by Plaintiff has suffered individual damages, including but not limited to economic harm and loss of consortium due to the injuries caused to Plaintiff, and will continue to suffer said damages, harm and loss of consortium in the future.

123. Defendant's conduct as alleged in this Complaint shows that Defendant acted maliciously, with aggravated or egregious fraud, and/or intentionally disregarded Plaintiff's rights, so as to warrant the imposition of punitive damages.

124. As a direct and proximate result of the defective condition of Lexapro® as manufactured by Defendants, Plaintiffs suffered injuries and damages as described below.

COUNT VII
Strict Products Liability Due To Inadequate Warning O.R.C. § 2307.76

125. Plaintiff Bossetti brings this cause of action in alternative to her common law claims.

126. Plaintiff incorporates the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

127. Defendant is the manufacturer, designer, marketer, distributor, and seller of Lexapro®.

128. It was reasonably foreseeable that women of childbearing years, such as Plaintiff would become pregnant while on Lexapro®, and that a very high percentage of children exposed to Lexapro® in utero would suffer devastating effects as a result.

129. The Lexapro® manufactured, designed, marketed, distributed and sold by Defendant was defective due to inadequate warning or instruction pursuant to O.R.C. § 2307.76, because at the time it left the control of Defendants and was supplied to Plaintiff, Defendant knew or should have known that their product was unreasonably dangerous as confirmed by the extensive body of published literature and its own internal data, because Lexapro® substantially and significantly increases the risk of developmental defects compared to other treatment options for treatment of depression.

130. Despite the fact that Defendant knew or should have known about the increased risk of defects with Lexapro® as compared to other treatment options for depression, Defendant failed to exercise reasonable care to adequately warn of the increased risk. In fact, Defendant denied in the Lexapro® product label at the time of Plaintiff's product use that the association between Lexapro® and developmental defects was causal.

131. The Lexapro® manufactured and supplied by Defendant was defective due to inadequate warning or instruction pursuant to O.R.C. § 2307.76, because at the time it left the control of Defendant and was supplied to Plaintiff, Defendant knew or should have known that their product was unreasonably dangerous, as confirmed by the extensive body of published literature and its own internal data, because higher doses of Lexapro® substantially and significantly increases the risk of teratogenic effects compared to lower doses.

132. The Lexapro® manufactured and supplied by Defendant was defective due to inadequate warning or instruction pursuant to O.R.C. § 2307.76, because at the time it left the control of Defendant and was supplied to Plaintiff, Defendant knew or should have known that their product was unreasonably dangerous, as confirmed by the extensive body of published literature and its own internal data, because ingestion of Lexapro® substantially and significantly increases the risk of harm compared to other medications.

133. Despite the fact that Defendant knew or should have known about the increased risk of harm with use of Lexapro®, Defendant failed to exercise reasonable care to adequately warn of the increased risk. In fact, Defendants made no reference in the Lexapro® product label to increased risk of developmental delays when used by pregnant women.

134. The Lexapro® manufactured and supplied by Defendants was defective due to inadequate warning or instruction pursuant to O.R.C. § 2307.76, because at the time it left the control of Defendant and was supplied to Plaintiff, Defendant knew or should have known that their product was unreasonably dangerous, as confirmed by the extensive body of medical literature and Defendant's internal data, because ingestion of Lexapro® substantially and significantly increases the risk of impaired cognitive function, neurodevelopmental delay, and behavioral disorders

135. Despite the fact that Defendant knew or should have known about the increased risk of impaired cognitive function, neurodevelopmental delay, and behavioral disorders caused by in utero exposure to Lexapro®, Defendant failed to exercise reasonable care to adequately warn of this increased risk. In fact, Defendants made no reference to any such increased risk in the Lexapro® product label at the time of Plaintiff's product use.

136. The Lexapro® manufactured and supplied by Defendants was defective due to inadequate warning or instruction pursuant to O.R.C. § 2307.76, because at the time it left the control of Defendant and was supplied to Plaintiff, Defendant knew or should have known that their product was unreasonably dangerous for any use by women of childbearing years, as confirmed by the extensive body of medical literature and Defendant's internal data, because ingestion of Lexapro® substantially and significantly increases the risk of harm to the developing fetus.

137. Despite the fact that Defendant knew or should have known that Lexapro® should warn specifically for women of childbearing years, Defendant failed to exercise reasonable care to adequately warn of the necessity of prohibiting women of childbearing years from ingesting this drug. Instead, Defendants denied that a cause and effect relationship had been proven between Lexapro® and developmental and other defects and advised that the benefits and the risks of Lexapro® should be weighed (based upon the inaccurate and incomplete information contained in the product label), without revealing that other drugs offered safer alternatives for women of childbearing years.

138. The Lexapro® manufactured and supplied by Defendant was also defective due to inadequate warning or instruction pursuant to O.R.C. § 2307.76, because at the time it left the control of Defendants and was supplied to Plaintiff, Defendant knew or should have known

that their product was unreasonably dangerous for any use by women of childbearing years, as confirmed by the extensive body of medical literature and Defendant's internal data.

139. Despite the fact that Defendant knew or should have known that using Lexapro® during pregnancy created a substantially greater risk to the fetus, Defendant failed to exercise reasonable care to adequately warn women and their doctors of the unreasonable risk posed by use of Lexapro® during pregnancy.

140. The Lexapro® manufactured and supplied by Defendant was also defective pursuant to O.R.C. § 2307.76 due to inadequate post-marketing warning or instruction, because after Defendant knew or should have known of the substantially increased risks as described above, Defendant failed to provide adequate post-market or post-approval warnings to consumers and/or their health care providers, which they have authority to do as the holder of the NDAs, and failed to revise the Lexapro® label to warn of the serious and substantially increased risk of fetal harm caused by Lexapro® as compared to other anti-depressant medications; nor did Defendants warn Plaintiff or her physician that alternative safer options were available and that Lexapro® should not be ingested by women of childbearing years.

141. The significantly increased risk of harm of Lexapro® is not an open and obvious danger or a matter of common knowledge.

142. As a direct and proximate result of Plaintiff's use of Lexapro® as manufactured, designed, marketed, distributed and sold by Defendant, Plaintiff suffered damages, including but not limited to personal injury, bodily harm, emotional distress, pain and suffering, permanent physical, mental, neurologic, cognitive and behavioral injuries, loss of enjoyment of life, economic and non-economic damages, and will continue to suffer suchinjuries, distress, pain and suffering, harm, damages, and economic loss in the future.

143. In addition, as a direct and proximate result of Plaintiff's use of defective Lexapro®, Plaintiff has suffered damages, including but not limited to economic harm and loss of consortium due to the injuries caused to S.R.B. , and will continue to suffer said damages, harm and loss of consortium in the future.

144. Defendant's conduct as alleged in this Complaint shows that Defendant acted maliciously, with aggravated or egregious fraud, and/or intentionally disregarded Plaintiff's rights, so as to warrant the imposition of punitive damages.

COUNT VIII
Strict Products Liability Nonconformance with Representations
O.R.C. § 2307.77

145. Plaintiff Bossetti brings this cause of action the alternative to her common law claims.

146. Plaintiff incorporates the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

147. At the time Defendant manufactured, designed, marketed, distributed and sold Lexapro® to Plaintiff, Defendant represented to consumers and the medical community through the product label that the benefits of Lexapro® in treating depressive disorders could outweigh the risk even for women of childbearing years.

148. However, as described herein, Defendant's Lexapro® failed to conform to these representations and instead is completely unacceptable for use among women of childbearing years, because the risk of fetal harm with Lexapro® is so high and so many safer options are available to treat depression and depressive disorders.

149. However, as described herein, Defendant's Lexapro® failed to conform to these representations because it in fact does causally increase the risk of birth defects, and the increase cannot be attributed to poor methodology, or genetics.

150. The failure of Lexapro® to conform to the representations made by Defendant in the product label, as described above, render the product defective pursuant to O.R.C. § 2307.77.

151. As a direct and proximate result of Plaintiff's use of defective Lexapro®, which failed to conform to manufacturer representations as described above, Plaintiff suffered damages, including but not limited to personal injury, bodily harm, emotional distress, pain and suffering, permanent physical, mental, neurologic, cognitive and behavioral injuries, loss of enjoyment of life, economic and non-economic damages, and will continue to suffer such injuries, distress, pain and suffering, harm, damages, and economic loss in the future.

152. In addition, as a direct and proximate result of Plaintiff's use of defective Lexapro®, Ms. Bossetti has suffered individual damages, including but not limited to economic harm and loss of consortium due to the injuries caused to their son S.R. Bossetti., and will continue to suffer said damages, harm and loss of consortiumin the future.

153. Defendant's conduct as alleged in this Complaint shows that Defendant acted maliciously, with aggravated or egregious fraud, and/or intentionally disregarded Plaintiff's rights, so as to warrant the imposition of punitive damages.

COUNT IX
Violation of N.J. Stat. § 2A:58C-2

154. Plaintiff DiMeglio brings this cause of action in the alternative to her common law claims and incorporates the preceding paragraphs herein.

155. For the reasons stated above, Lexapro was not reasonably fit, suitable or safe for its intended purpose because it deviated from the design specifications, formulae, or performance

standards of the manufacturer or from otherwise identical units manufactured to the same manufacturing specifications or formulae, specifically in that the design of the product included a product label which indicated Lexapro's benefits outweighed the risks to pregnant mothers and women of childbearing years.

156. Further, Lexapro was defective because it failed to contain adequate warnings or instructions and was designed in a defective manner.

Upon information and belief, Defendant deliberately concealed the causal relationship between in utero exposure to Lexapro or other SSRI's and neurodevelopmental delay or failed to disclose this relationship despite knowledge of these harmful effects. Further, Defendant chose to rely on the postmarket regulatory process as a basis to fail to disclose this relationship.

DAMAGES AND PRAYER FOR RELIEF

157. Plaintiffs incorporate the allegations contained in the foregoing paragraphs as if fully set forth in the following paragraphs.

158. The facts set out above demonstrate that, as a direct and proximate result of Defendant's conduct, Plaintiffs have each suffered severe economic and non-economic losses and injuries for which they are entitled to recover damages, including without limitation the following damages in excess of $75,000 per plaintiff without the need for any aggregation of damages:

(a) bodily injury, conscious pain, suffering, mental anguish, mental suffering, embarrassment, shame, loss of enjoyment of life, shortened life expectancy, death, loss of association, loss of earnings, loss of profits, loss of salary;

(b) the reasonable and necessary expenses for the medical treatment rendered to Plaintiff in the past and that will be medically probable in the future;

(c) compensation for Plaintiff's permanent mental and behavioral impairment;

(d) punitive damages

(e) future economic damages during the age of minority and beyond the age of 18, including lost wages of Plaintiff;

(f) costs of this suit;

(g) reasonable attorneys' fees;

(h) all other actual damages available under applicable law;

WHEREFORE, Plaintiff prays that the Court:

(a) Enter a judgment against the Defendant,

(b) Enter a judgment against Defendant for compensatory damages.

(c) Enter a judgment against Defendant for costs and pre- and post-judgment interest.

(d) Require such other and further relief as the Court deems just and proper under the circumstances.

Respectfully submitted,

/s/ Andrew S. Baker
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DEMAND FOR JURY TRIAL

Plaintiffs hereby demand a trial by jury on all issues.

/s/ Andrew S. Baker
Andrew S. Baker (0080196)
Attorney for Plaintiff